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Overview of tremor

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INTRODUCTION

Tremor is defined as an involuntary, rhythmic, and oscillatory movement of a body part [1]. It is caused by either alternating or synchronous contractions of antagonistic muscles. Tremor is the most common of all movement disorders, occurring from time to time in many normal individuals in the form of exaggerated physiologic tremor [2].

This topic will provide an overview of the classification, clinical features, diagnostic evaluation, and treatment of tremor, including essential tremor (ET), which also is discussed in greater detail separately. (See "Essential tremor: Clinical features and diagnosis" and "Essential tremor: Treatment and prognosis" and "Surgical treatment of essential tremor".)

CLASSIFICATION

Tremors have been variably defined and categorized over time, and classification is made difficult by overlapping clinical characteristics and etiologies. The most common distinction is based on the activating conditions (ie, at rest versus action), but other clinical characteristics, such as frequency and body distribution, also guide the evaluation and etiologic diagnosis.

A clinical and etiologic classification scheme has been proposed by the International Parkinson and Movement Disorder Society [1]. It provides a framework upon which currently recognized and new tremor syndromes can be defined.

Clinical characteristics — Clinical classification of tremor is based on history, tremor characteristics, associated neurologic and systemic signs, and, in some cases, additional testing.

Rest versus action — The activating conditions that give rise to a tremor are a key distinguishing feature (table 1).

Rest tremor occurs in a body part that is fully supported, relaxed, and not voluntarily activated.

Action tremor occurs with voluntary muscle contraction and is further subdivided into three types:

- Kinetic tremor occurs during any voluntary movement. Kinetic tremors include:
  - Simple kinetic tremor, in which tremor is roughly the same throughout the course of a voluntary movement
  - Intention tremor, characterized by a crescendo increase in tremor as the affected body part approaches its target
  - Task-specific kinetic tremor, which occurs during a specific task such as writing
- Postural tremor occurs when a specific posture or position, such as holding the arms outstretched or while standing, is voluntarily maintained.
- Isometric tremor occurs during muscle contraction against a stationary object, such as making a fist or squeezing an object.

Tremor frequency and body distribution — Tremor frequency (oscillations per second) is used to describe tremor, although it is not particularly helpful in diagnosis as most pathologic tremors have a frequency of 4 to 8 Hertz (Hz) [1]. The most common subdivisions used for descriptive purposes are <4, 4 to 8, 8 to 12, and >12 Hz. Tremor frequency is typically estimated clinically but can be accurately measured using a motion transducer or electromyography (EMG).

The anatomic distribution of tremor can be classified as:

- Focal (only one body region is affected, such as voice, head, jaw, or one limb)
- Segmental (two or more contiguous body parts in the upper or lower body are affected, such as head and arm)
- Hemitremor (one side of the body is affected)

- Generalized (upper and lower body are affected)

Isolated versus combined — Tremor syndromes are specific combinations of clinical signs and symptoms that commonly coexist. Syndromes can help narrow the search for an etiologic diagnosis. Syndromes may be **isolated**, when tremor is the only neurologic manifestation, or **combined**, when other systemic or neurologic signs coexist with the tremor (table 2) [1]. Importantly, syndromes may have multiple etiologies, and a particular etiology may produce multiple clinical syndromes.

Etiology — A vast number of diseases, disorders, medications, toxins, and substances cause tremor. Etiologies can be broadly classified as genetic, acquired, and idiopathic.

Some of the more common etiologies of rest and action tremors are listed in the table (table 3). In addition to these, there are many uncommon or rare conditions that may be associated with tremor; most are action tremors [3].

REST TREMORS

Parkinson disease (PD) and other parkinsonian syndromes are the most common causes of rest tremor (algorithm 1).

General features — Rest tremor is evident with the affected body part fully supported against gravity and completely at rest. It is temporarily dampened or abolished during voluntary activity. Rest tremors usually fluctuate in amplitude and may appear and disappear depending upon the degree of patient repose, whether the patient feels that they are under observation, and other factors, including stress, excitement, or recent exercise.

Rest tremor is typically less disabling than postural or kinetic tremors because of its absence during voluntary activity. However, a rest tremor may quickly reappear as soon as the body part assumes a new resting or antigravity posture (a phenomenon referred to as "re-emergent tremor"), and in this case will interfere with the use of eating utensils, handwriting, typing, and other purposeful postures or movements. Rest tremor with these postural features is more of a handicap or disability than pure rest tremor, which is of mainly cosmetic concern.

Dystonic tremor is usually postural or task specific but it can occur at rest, in which state it tends to be jerky and irregular, unlike the smoother, more rhythmic pattern of the tremor of parkinsonism. (See 'Dystonic tremor' below.)

On examination, when rest tremor is absent or minimally apparent, it usually can be activated or enhanced by having the patient walk, perform repetitive movements of the opposite limb, or

perform a cognitive task, such as naming the months of the year backwards.

Idiopathic Parkinson disease — The rest tremor in PD typically appears first in one hand or less commonly in one leg. As the disease progresses, tremor may spread from one hand to involve the ipsilateral leg and/or the contralateral arm. Tremor of the leg or foot is more commonly due to PD than to essential tremor (ET). The face, lips, and jaw may be involved but, unlike ET or cerebellar disease, PD only occasionally produces tremor of the entire head. (See "Clinical manifestations of Parkinson disease", section on 'Tremor'.)

When the tremor in PD is limited to distal muscles of the hand, it may produce a characteristic "pill-rolling" pattern, with a frequency of 4 to 6 Hertz (Hz). With increasing severity, the tremor may become more continuous, larger in amplitude, and more proximal in distribution, but the frequency remains constant.

Postural tremor may coexist with rest tremor or may be present by itself in some patients with PD, sometimes leading to an incorrect diagnosis of ET. (See 'Essential tremor' below.)

Tremor-dominant Parkinson disease — Patients with Parkinson disease can go many years into their disease with tremor as the only or the most prominent sign of their disorder. When this happens, this is referred to as "tremor-dominant PD," although this designation is most reliably applied in retrospect, when the relatively benign course has already been charted.

Isolated rest tremor (SWEDD) — The radiographic term "SWEDD" (Scans Without Evidence of Dopaminergic Deficit) has been used to designate patients with relatively isolated rest and action tremors of the arms, resembling those of early PD, that have failed to evolve over time into more generalized PD [4]. Unlike patients with PD, these individuals lack evidence for nigrostriatal dopamine deficiency with dopamine transporter imaging (DaTscan). Patients with SWEDD sometimes exhibit reduced arm swing and mild focal dystonia on the affected side, and may have jaw or head tremor or facial hypomimia, but no signs of parkinsonian bradykinesia [4]. They are therefore to be distinguished from individuals with tremor-dominant PD. (See "Diagnosis and differential diagnosis of Parkinson disease", section on 'Scans without evidence of dopaminergic deficit (SWEDD)'.)

Others — Other disorders associated with rest tremor include drug-induced parkinsonism secondary to dopamine receptor blocking agents; the atypical parkinsonian syndromes such as dementia with Lewy bodies, multiple system atrophy, and progressive supranuclear palsy; Wilson disease; non-Wilsonian hepatocerebral degeneration; and thalamic or midbrain injury due to stroke, trauma, or demyelinating disease. Rest tremor may also occur as a "spillover" phenomenon in a variety of disorders in which very severe action tremors predominate, such as

Wilson disease, multiple sclerosis, severe forms of ET [5], and other cerebellar or extrapyramidal syndromes.

Rubral tremor and dystonic tremor are combined tremor syndromes in which the tremor can have a rest component but is often mixed with postural and kinetic features as well. (See 'Rubral tremor' below and 'Dystonic tremor' below.)

ACTION TREMORS

Action tremors are the largest group of tremors. They include postural tremors, elicited during examination with the arms suspended against gravity in a fixed posture, and kinetic tremors, which emerge during the course of goal-directed activity (eg, drinking from a glass of water, pouring water from one cup into another, writing, and drawing of a spiral).

Physiologic tremor — Normal individuals have a very low-amplitude, high-frequency physiologic tremor of approximately 10 to 12 Hertz (Hz). Physiologic tremor is usually not visible under ordinary circumstances, although some people have a natural proclivity to demonstrate mild, nondisabling physiologic tremulousness. Many factors can enhance the tremor to the point of detection, most often by increasing sympathetic activity [2].

- **Medications** – Common drugs that increase adrenergic activity include beta-adrenergic agonists such as terbutaline, albuterol, isoproterenol, epinephrine, amphetamines, selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants, levodopa, nicotine, and xanthines such as theophylline and caffeine (table 4).

- **High adrenergic state** – Anxiety, excitement, fright, muscle fatigue, hypoglycemia, alcohol and opioid withdrawal, thyrotoxicosis, fever, and pheochromocytoma also enhance adrenergic activity. (See "Neurologic manifestations of hyperthyroidism and Graves' disease", section on 'Tremor'.)

Enhancement of physiologic tremor is the most common cause of action tremor. Thus, a medical rather than primary neurologic cause for action tremor should be considered first in most cases.

Drug-induced tremor — Drugs and toxins that commonly increase physiologic tremor or induce action tremor by other means include lithium, antidepressants, corticosteroids, sodium valproate, tacrolimus, bromides, mercury, lead, and arsenic (table 4).

Essential tremor — Essential tremor (ET) is reviewed here briefly and is discussed in detail separately. (See "Essential tremor: Clinical features and diagnosis" and "Essential tremor:

Treatment and prognosis" and "Surgical treatment of essential tremor".)

ET is the most common neurologic disorder that causes action tremor, with an estimated prevalence worldwide of up to 5 percent of the population. The incidence of ET increases with age, although it often affects middle-aged individuals, especially when it is familial. A family history is present in 30 to 70 percent of cases, and evidence suggests an autosomal dominant pattern of inheritance with reduced penetrance. (See "Essential tremor: Clinical features and diagnosis", section on 'Pathogenesis and genetics'.)

ET varies from a low-amplitude, high-frequency postural tremor of the hands to a much larger-amplitude, and at times lower-frequency, postural and action tremor that is activated by particular postures and actions. ET most often affects the hands and arms and can be asymmetric. Less often, it may involve the head, voice, trunk, and legs. ET becomes immediately apparent in the arms when they are held outstretched; it typically increases at the very end of goal-directed movements such as drinking from a glass or finger-to-nose testing. Tremor in the legs is unusual in ET. Tremor of the head may be vertical ("yes-yes") or horizontal ("no-no") and is usually associated with tremor of the hand or voice. ET is typically relieved by small amounts of alcohol. Caffeine can worsen the tremor, although this effect is much more commonly seen with physiologic tremor.

The diagnosis of ET is based upon clinical features (table 5). (See "Essential tremor: Clinical features and diagnosis", section on 'Diagnosis'.)

The main considerations in the differential diagnosis of ET are enhanced physiologic tremor, drug-induced tremor, parkinsonian tremor, tremor caused by cerebellar disorders, and dystonic tremor (algorithm 1).

A frequent entity on the differential diagnosis of ET is parkinsonian tremor (table 6 and table 7 and table 8). Differentiation from classic rest tremor should be straightforward; however, some patients with Parkinson disease (PD) also have an action tremor indistinguishable from ET. In fact, it is not unusual for patients with PD to present with a postural tremor before they display other signs of PD. Likewise, patients with severe ET may have a rest component to their tremor, and some patients with ET exhibit mild nonprogressive parkinsonism.

Primary writing tremor — Many action tremors are particularly severe during the act of writing. Tremor that occurs exclusively while writing, and not during other voluntary motor activities, is referred to as "primary writing tremor" [6]. This tremor is limited to the hand and causes relatively large-amplitude supination-pronation movements at a frequency of 5 to 6 Hz. The low frequency and large amplitude of the tremor, its frequent occurrence in writer's

"cramp" or writer's dystonia, its relative resistance to propranolol, and its occasional response to anticholinergic drugs suggest a closer relationship to dystonia than to ET. (See "Etiology, clinical features, and diagnostic evaluation of dystonia", section on 'Task-specific dystonia'.)

Orthostatic tremor — Orthostatic tremor is a postural tremor limited to the legs and trunk that occurs exclusively while standing [7-10]. In two reports of 184 and 68 patients, there was a female predominance (64 and 77 percent, respectively) [11,12]. The mean age of onset was between 54 and 59 years (range 13 to 85 years). Both high- and low-frequency orthostatic tremors have been described; their relationship to ET is uncertain. In cases of high-frequency tremor, movements of the legs may be so low in amplitude that they initially escape clinical detection.

Cerebellar tremor — "Cerebellar tremor" is the term used to describe a tremor that is caused by known cerebellar pathology. It can be postural, simple kinetic, intention, or any combination of these features. In severe cases, cerebellar tremor, like ET, can spill over and also occur at rest. Tremor frequency is typically low (3 to 4 Hz) and can be associated with ataxia and dysmetria.

Cerebellar tremor is due to disturbances anywhere along the path of the cerebellar outflow projection system from the dentate nucleus of the cerebellum to its termination in the ventral lateral nucleus of the thalamus. The most common causes are multiple sclerosis, midbrain trauma, and stroke (table 3). Degenerative and inflammatory diseases of the cerebellum, severe forms of ET, Wilson disease, hepatocerebral degeneration, and mercury poisoning may also produce this tremor.

Cerebellar tremor typically has a component of intention tremor in that the tremor increases in severity, either gradually or abruptly, as the hand moves closer to its target. Intention tremors are usually very large in amplitude due to involvement of proximal muscles and are sometimes difficult to distinguish from severe cerebellar ataxia. The frequent association with ataxia, dysmetria, titubation, and other cerebellar signs serves to identify the cerebellar origin of intention tremor.

Titubation of the head and neck ("to and fro" movements) may be associated with cerebellar tremor; it is distinguished from essential head tremor by the presence of other cerebellar findings.

Rubral tremor — Rubral tremor, sometimes called Holmes tremor after the early 20th century English neurologist who described it, is a subtype of cerebellar tremor with a greater degree of proximal, high-amplitude movement. Rubral tremor is caused by midbrain, thalamic, cerebellar, or pontine injury [13]. In many cases of rubral tremor, there are combined lesions that interrupt

the outflow pathway from the cerebellum to the motor thalamus, often involving the superior cerebellar peduncle, substantia nigra, or red nucleus.

Rubral tremor is present at rest but tends to have a slower frequency (3 to 5 Hz) than typical Parkinson rest tremor (5 to 7 Hz). It also persists unchanged or increases with postural changes or goal-directed activity. It may produce a combination of rest, postural, simple kinetic, and intention tremors.

Neuropathic tremor — Postural or kinetic tremors are sometimes associated with large fiber peripheral neuropathy. This association is most commonly observed in hereditary neuropathies, during the recovery phase of some cases of Guillain-Barré syndrome, and in chronic inflammatory demyelinating polyneuropathy (CIDP). Muscle weakness and loss of proprioceptive or muscle spindle inputs may account for the tremor. The frequency and amplitude of neuropathic tremor may vary greatly when associated with a proprioceptive deficit. (See "Overview of polyneuropathy".)

Dystonic tremor — Dystonic tremor refers to tremor in a body part that is affected by dystonia. Because of the association with dystonia, dystonic tremor is an example of a combined tremor syndrome [1]. Common examples include dystonic head tremor and segmental tremulous dystonia of the head and upper limbs, which occur due to cervical dystonia. (See "Etiology, clinical features, and diagnostic evaluation of dystonia".)

Dystonic tremors are usually postural or task specific but sometimes occur at rest, in which state they tend to be jerky and irregular. The description of an alleviating maneuver (geste antagoniste, sensory trick) can be helpful in distinguishing dystonic tremor from other tremor syndromes, such as ET. Exacerbation of dystonic tremor by an attempt to maintain certain postures can also be helpful in the examination.

FUNCTIONAL TREMORS

Functional tremor, previously known as psychogenic tremor, is typically characterized by complex rest, postural, and kinetic tremor with abrupt onset, a static course, changeable features, functional disability out of proportion to tremor magnitude, and resistance to treatment. Any body part may be involved, but, remarkably, the fingers are often spared, with much of the tremor of the arm occurring at the wrist.

Other features of functional movement disorders are often present, such as distractibility, variability of symptoms, and clinical features that are incongruous with known tremor syndromes. Examination usually shows variable tremor direction and frequency, distractibility

(attenuation or elimination of tremor with distraction maneuvers such as repetitive tapping tasks with an uninvolved opposite hand or foot), and/or tremor entrainment (ie, shift of tremor frequency to the speed of a distraction maneuver) (movie 1 and movie 2). (See "Functional movement disorders", section on 'Clinical features'.)

EVALUATION

The diagnostic approach to patients with tremor involves the history, physical examination, and selected laboratory studies [14]. Action tremor is most common and, of these, essential tremor (ET) and enhanced physiologic tremor are the most frequent causes (algorithm 1) [15]. Patients with tremor due to other disorders such as hyperthyroidism, Parkinson disease (PD), dystonia, Wilson disease, or medications frequently have additional signs or symptoms that help point to the diagnosis, although this is not always the case. The criteria for ET (table 5) and methods for distinguishing it from other tremors are shown in the tables (table 1 and table 3 and table 6 and table 7 and table 8).

History — The history concerning the age of onset and evolution of tremor is usually straightforward, since it is a highly visible symptom that is readily evident to the patient and family members. Examination of previous handwriting samples may be useful in determining the precise time of onset.

Patients should be asked specific questions about:

- **What parts of the body are affected** – Body parts that are affected and symmetry can be helpful clues to the likely diagnosis. Head tremor is common in ET but unusual in PD; conversely, jaw tremor and rest tremor in the leg are common in PD and rare in ET. Jaw or chin tremor at rest should be distinguished from lower lip tremor that occurs with activation, as facial tremor with activation may be seen in ET. Vocal tremor is strongly suggestive of ET. Asymmetry or unilaterality is typically more characteristic of PD tremor than ET. Any involuntary posturing that occurs with tremor should raise the possibility of a dystonic tremor.

- **Precipitating, aggravating, or relieving factors –** Patients should be asked about the effects of caffeine, alcohol, medications, exercise, fatigue, or stress on their tremor; a complete list of all medications should be reviewed to exclude the possibility of enhanced physiologic tremor and drug-induced tremor (see 'Physiologic tremor' above). Mitigation of tremor by alcohol is classically described in ET but is not highly sensitive or specific.

- **Associated neurologic symptoms** – Patients should also be questioned about any associated neurologic symptoms that would assist in diagnosis, including loss of dexterity, stiffness, stooped posture, voice softness, shuffling gait, acting out of dreams (all suggestive of PD), cognitive changes, and visual hallucinations (suggestive of dementia with Lewy bodies). Lack of any of the above features along with a predominantly action tremor would be suggestive of ET.

- **Family history** – Family history in ET reflects an autosomal dominant pattern of inheritance in approximately half of all patients. Parkinsonian tremor is usually sporadic, but a family history of PD is present in approximately 15 percent of cases; the disease affects first-degree relatives (parents, siblings, or offspring) in approximately half of the familial cases. Autosomal PD affecting three or more generations is rare.

Examination — A detailed neurologic examination is important to identify specific features of the tremor (including its frequency, amplitude, pattern, and body distribution), activating conditions, and other neurologic findings, if present [1,16].

Examination begins with observations of the tremor during the interview and continues throughout the patient visit. Due to stress, many patients with tremor are more symptomatic during the early part of the interview or examination than after they become acclimated to the clinical encounter.

A focused examination for tremor includes the following maneuvers, which supplement the basic neurologic examination:

- Observe the limbs at rest, fully supported by gravity

- Observe the limbs with posture (elevated against gravity) in an outstretched position and in a wing-beating position (for the arms)

- Test for distractibility or entrainment while in an outstretched posture

- Test finger-to-nose and heel-to-shin movements

- Obtain a writing sample and spirals for each hand (with arm unsupported)

- Observe the patient pouring water from one cup to another

Clues to the type and etiology of tremor arise at all stages of the examination, and the body parts involved narrow the differential:

- **Head or facial involvement** – Patients with head tremor should be observed closely for tremor in other body parts. Horizontal or vertical head tremor is usually associated with tremor elsewhere, commonly in the arms and/or voice, and these findings suggest ET. Isolated head tremor should raise the possibility of cervical dystonia or midline cerebellar syndromes.

  Localized jaw tremor is more commonly a manifestation of parkinsonism, while localized face or lip tremor may occur in ET. Essential voice tremor is readily audible and may be further enhanced by having the patient hold a prolonged note.

- **Upper extremity involvement** – Tremor in the arm is observed with the affected limb fully supported on the patient's lap or the arm of a chair, with the limb elevated against gravity, and during goal-directed movements. These maneuvers are aimed at determining whether the tremor is predominantly a rest or an action tremor.

  Rest tremor characteristic of parkinsonism may not appear unless the arm muscles are fully relaxed and supported against gravity. Maneuvers that activate rest tremor include repetitive movements of the opposite hand, walking, and mental distraction such as reciting the months of the year backwards. The hands and arms should be observed closely during these activities for an increase in tremor, which is suggestive of parkinsonism rather than ET.

  Postural and kinetic tremors are best elicited with the arms held outstretched; with the shoulders abducted, elbows flexed, and index fingers held an inch apart in front of the face (to elicit any "wing-beating" tremor); during finger-to-nose maneuvers; and while drinking from or pouring from a paper cup.

  Writing and drawing may demonstrate the large, tremulous, angulated loops of ET or the micrographia of parkinsonism. A drawing sample of a spiral is commonly used to evaluate ET severity and can be repeated to be compared over time. Having the patient draw a spiral with the hand suspended above the table increases the sensitivity of this test.

  While examining for postural tremor and kinetic tremor, careful observation for abnormal posturing of the tremulous fingers, hand, or upper limb is required to rule out a dystonic tremor. Dystonic posturing can also be elicited during examination of repetitive movements of the opposite limb, writing, and walking.

- **Lower extremity involvement** – Tremor of the leg should be assessed with the limb at rest, during heel-to-shin testing, and while standing and walking. Leg tremor is more commonly due to parkinsonism than ET.

Functional tremor can involve any body part and may be suspected when tremor frequency, amplitude, and/or direction varies from moment to moment. Functional tremors may become more irregular or subside entirely when the patient is distracted by performing a complex, repetitive motor task with the contralateral limb. The tremor may also entrain to the frequency of the repetitive movement being tested in another limb. For this reason, it can be helpful to test for entrainability by asking patients to tap their fingers at the same rhythm as the examiner, and then slowly increase or decrease the frequency of the taps, looking for associated changes in the rhythm of the tremor. (See 'Functional tremors' above.)

The gait is almost always normal in patients with ET, while it is characteristically narrow-based and shuffling in PD and wide-based and ataxic in cerebellar disorders. The gait may have nonphysiologic qualities in patients with functional tremors.

Laboratory studies — The routine laboratory evaluation of tremor should include tests of thyroid function, diagnostic studies to exclude Wilson disease, and, rarely, screening for heavy metal poisoning such as mercury or arsenic if an environmental cause is suspected. Wilson disease should be suspected in anyone under age 40 who has tremor or other involuntary movements or postures [2] (see "Wilson disease: Clinical manifestations, diagnosis, and natural history"). Hypoglycemia and pheochromocytoma may need to be ruled out in patients with enhanced physiologic tremor.

Brain imaging is usually not indicated in patients with classic presentations of ET, PD, enhanced physiologic tremor, or drug-induced tremor, although normal pressure hydrocephalus (NPH) or stroke can rarely produce a parkinsonian phenotype. In cases where there is clinical uncertainty as to whether a patient has ET or isolated PD tremor, striatal dopamine transporter imaging (DaTscan) will detect striatal dopamine deficiency even very early in PD but will be normal in ET. (See "Diagnosis and differential diagnosis of Parkinson disease".)

Magnetic resonance imaging (MRI) can be useful in patients thought to have rubral or Holmes tremor, which is typically due to a lesion in the cerebellar outflow network; in those with Wilson disease; and in those with a pure hemidystonic tremor, which may be due to a contralateral structural lesion.

Quantitative computerized analysis of tremor is available in some tertiary care facilities, but its ability to reliably distinguish between tremor types has not been established.

TREATMENT

The treatment of tremor is symptomatic, and some forms lack any useful pharmacologic therapy. Even medications known to have some benefit for certain tremor syndromes usually have only partial effect. For combined tremor syndromes, treatment of the underlying disorder is the primary approach.

Physical and occupational therapy may be of benefit to help identify coping mechanisms and compensatory strategies. In patients with refractory or disabling tremor, the approach should be individualized according to the underlying cause. In addition to medications, some patients may be candidates for surgical therapies.

- **Rest tremor** – Rest tremor associated with Parkinson disease (PD) or other parkinsonian disorders is managed by treatment of the underlying disorder, typically with antiparkinson agents such as carbidopa-levodopa, amantadine, dopamine agonists, and, less commonly, anticholinergic drugs and zonisamide. Deep brain stimulation (DBS) is an option for some patients with refractory tremor due to PD, but not due to other parkinsonian disorders. This topic is discussed separately. (See "Initial pharmacologic treatment of Parkinson disease" and "Device-assisted and lesioning procedures for Parkinson disease".)

- **Enhanced physiologic tremor** – Physiologic tremor is best managed by reduction or removal of the responsible offending medication or toxin (table 4); diagnosis and treatment of possible associated endocrine disorders; and dealing with stress, anxiety, or fatigue (see 'Physiologic tremor' above). Single doses of propranolol, a short-acting beta blocker, taken in anticipation of social situations that are likely to exacerbate tremor (eg, as with tremor associated with public speaking or having dinner with friends) are useful in some patients.

- **Essential tremor (ET)** – ET can be treated pharmacologically with partial symptomatic benefit. Propranolol and primidone are the preferred, evidence-based drugs to use as initial monotherapy, and when used in combination they can sometimes offer additive benefit (algorithm 2). Second-line medications for ET include agents such as topiramate and gabapentin. Botulinum toxin injections can be effective for head tremor and vocal tremor in ET, and in selected cases for upper extremity tremor. Both DBS and unilateral thalamotomy, which can now be performed via focused ultrasound with MRI guidance, are effective for the treatment of medically refractory ET. (See "Essential tremor: Treatment and prognosis" and "Surgical treatment of essential tremor".)

- **Orthostatic tremor** – Orthostatic tremor involves the legs and trunk and occurs exclusively while standing (see 'Orthostatic tremor' above). In one retrospective case series of 184 patients with orthostatic tremor, treatment with benzodiazepines, mainly clonazepam, was associated with moderate or marked relief in approximately one-third of the patients [11].

Other classes, such as antiseizure medications (eg, gabapentin, primidone, valproate) were associated with lower rates of improvement, and lack of a placebo control was a major limitation. Limited evidence suggests that some patients with medically refractory orthostatic tremor show modest improvement with DBS of the ventral intermediate nucleus of the thalamus [17].

- **Cerebellar tremor** – Cerebellar tremors lack any useful pharmacotherapy. The rare patient with severe intention tremor and little or no ataxia, such as sometimes occurs in multiple sclerosis, may be helped by DBS of the ventral intermediate nucleus of the thalamus. (See "Surgical treatment of essential tremor".)

- **Rubral tremor** – Rubral tremor sometimes responds to levodopa therapy [13,18]. Other medications that may have some benefit are levetiracetam and trihexyphenidyl; thalamic DBS has been used with some success for medically refractory patients but requires more study [19].

- **Dystonic tremor** – Dystonic tremor of the head related to cervical dystonia can be improved with botulinum toxin injections into bilateral cervical muscles. If sternocleidomastoid muscles are injected bilaterally, small doses should be used to avoid dysphagia. Dystonic tremor of the limbs and jaw can also be improved with botulinum toxin injections into involved muscles. (See "Treatment of dystonia in children and adults", section on 'Focal dystonia' and "Cervical dystonia: Treatment and prognosis", section on 'Botulinum toxin injections'.)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Essential tremor".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Tremor (The Basics)" and "Patient education: Essential tremor (The Basics)")
- Beyond the Basics topics (see "Patient education: Tremor (Beyond the Basics)")

PATIENT PERSPECTIVE TOPICS

Patient perspectives are provided for selected disorders to help clinicians better understand the patient experience and patient concerns. These narratives may offer insights into patient values and preferences not included in other UpToDate topics. (See "Patient perspective: Essential tremor with onset in childhood" and "Patient perspective: An artist with essential tremor".)

SUMMARY AND RECOMMENDATIONS

- **Classification** – Clinical classification of tremor is based on history, tremor characteristics, associated neurologic and systemic signs, and, in some cases, additional testing. A vast number of diseases, disorders, medications, toxins, and substances cause tremor. Etiologies can be broadly classified as genetic, acquired, and idiopathic. (See 'Classification' above.)
- **Rest versus action** – The activating conditions that give rise to a tremor are a key distinguishing feature (table 1). Rest tremor occurs in a body part that is fully supported and not voluntarily activated, whereas action tremor occurs with voluntary muscle contraction. Action tremors can be further characterized as kinetic, postural, and isometric. (See 'Rest versus action' above.)
- **Causes of rest tremor** – Parkinson disease (PD) and other parkinsonian syndromes are the most common causes of rest tremor (table 2). (See 'Rest tremors' above.)
- **Causes of action tremor** – Examples of isolated tremor syndromes in which action tremor can be the sole neurologic symptom include (table 2):
  - Enhanced physiologic tremor (table 4) (see 'Physiologic tremor' above)
  - Essential tremor (ET) (table 5) (see 'Essential tremor' above)
  - Task-specific tremors (eg, writing tremor) (see 'Primary writing tremor' above)

- Orthostatic tremor (see 'Orthostatic tremor' above)

Examples of combined tremor syndromes in which action tremor may be seen in association with other neurologic or systemic signs and symptoms include:

- Cerebellar tremor (see 'Cerebellar tremor' above)
- Neuropathic tremor (see 'Neuropathic tremor' above)
- Dystonic tremor (see 'Dystonic tremor' above)
- Functional tremor (see 'Functional tremors' above)

- **Evaluation** – A detailed neurologic examination is important to identify specific features of the tremor (including its frequency, amplitude, pattern, and body distribution), activating conditions, and other neurologic findings, if present. (See 'Evaluation' above.)

  The routine laboratory evaluation of tremor should include tests of thyroid function, diagnostic studies to exclude Wilson disease, and, rarely, screening for heavy metal poisoning such as mercury or arsenic if an environmental cause is suspected. Brain imaging is usually not indicated in patients with classic presentations of ET, PD, enhanced physiologic tremor, or drug-induced tremor (algorithm 1). (See 'Laboratory studies' above.)

- **Treatment** – Physical and occupational therapy may be of benefit in patients with limb tremor to help identify coping mechanisms and compensatory strategies. Some forms of tremor are responsive to symptomatic pharmacotherapy. (See 'Treatment' above.)

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GRAPHICS

Activation classification of tremor

Tremor type	Definition	Clinical features and provocative maneuvers
Rest tremor		
	Occurs in a body part that is supported in such a way that skeletal muscle activation is neither necessary nor intended.	The patient is recumbent on a bed or seated on a chair with the body part supported. Tremor is often enhanced by the performance of cognitive tasks or motor tasks with other body parts, and i is often suppressed, at least temporarily by voluntary muscle contraction.
Action tremors		
Postural	Occurs in an attempt to maintain a specific posture or position against the force of gravity.	Extending the upper limbs horizontally, pointing at objects, sitting erect without support for the upper body, standing, protruding the tongue.
Kinetic (including simple kinetic, intention, and task-specific tremor)	Occurs during any voluntary movement. Intention tremor worsens as the body part approaches its target.	Finger-to-nose testing, heel-to-shin testing, reaching, writing, drawing, pouring water into a cup, eating with utensils, speaking. With task-specific tremor, perform the task associated with tremor.
Isometric	Occurs during a muscle contraction against a rigid, stationary object.	Pushing against a wall, flexing the wrist against a table, making a fist.

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Graphic 72091 Version 17.0

Tremor syndromes

Isolated tremor syndromes	Combined tremor syndromes	Other tremor syndromes
Action or rest tremors: ▪ Essential tremor ▪ Essential tremor plus Focal tremors: ▪ Voice tremor ▪ Head tremor ▪ Jaw tremor ▪ Face tremor ▪ Essential palatal tremor Task- and position-specific tremors: ▪ Writing tremor ▪ Sports tremor ▪ Musician's tremor Orthostatic tremors: ▪ Primary orthostatic tremor ▪ Pseudo-orthostatic tremor	▪ Dystonic tremors ▪ Parkinsonism-associated tremors ▪ Intention tremor (with cerebellar signs) ▪ Rubral (Holmes) tremor (with brainstem or cerebellar signs) ▪ Myorhythmia (with brainstem or cerebellar signs) ▪ Symptomatic palatal tremor	▪ Functional tremor ▪ Indeterminate tremor

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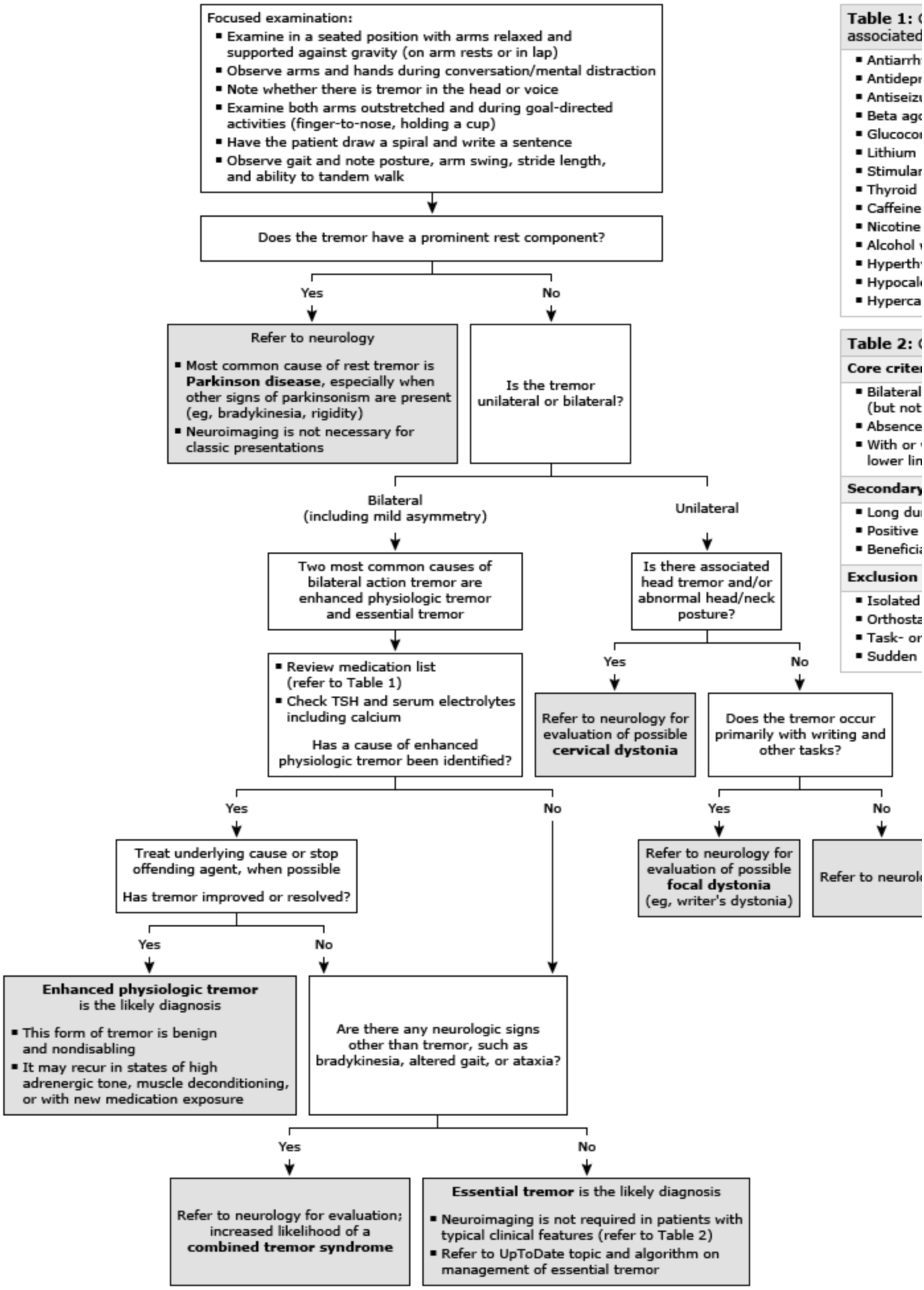
Common tremor etiologies

Resting tremor
Parkinson disease
Parkinsonian syndromes
Midbrain (rubral) tremor
Wilson disease
Severe essential tremor
Postural-action tremor
Enhanced physiologic tremors (eg, drug-induced and toxic tremors)
Essential tremor
Task-specific tremors
Other extrapyramidal disorders
Parkinson disease
Wilson disease
Dystonia
Cerebellar disease
Neuropathic tremor
Intention tremor (cerebellar outflow)
Cerebellar disease
Multiple sclerosis
Midbrain stroke
Midbrain trauma

Graphic 68810 Version 4.0

Practical approach to characterizing upper extremity tremor in adults in primary care


Focused examination:
Examine in a seated position with arms relaxed and supported against gravity (on arm rests or in lap)
Observe arms and hands during conversation/mental distraction
Note whether there is tremor in the head or voice
Examine both arms outstretched and during goal-directed activities (finger-to-nose, holding a cup)
Have the patient draw a spiral and write a sentence
Observe gait and note posture, arm swing, stride length, and ability to tandem walk
Does the tremor have a prominent rest component?
Yes
No
Refer to neurology
Most common cause of rest tremor is Parkinson disease, especially when other signs of parkinsonism are present (eg, bradykinesia, rigidity)
Neuroimaging is not necessary for classic presentations
Is the tremor unilateral or bilateral?
Bilateral (including mild asymmetry)
Unilateral
Two most common causes of bilateral action tremor are enhanced physiologic tremor and essential tremor
Review medication list (refer to Table 1)
Check TSH and serum electrolytes including calcium
Has a cause of enhanced physiologic tremor been identified?
Is there associated head tremor and/or abnormal head/neck posture?
Yes
No
Refer to neurology for evaluation of possible cervical dystonia
Does the tremor occur primarily with writing and other tasks?
Yes
No
Refer to neurology for evaluation of possible focal dystonia (eg, writer's dystonia)
Refer to neurol
Yes
No
Treat underlying cause or stop offending agent, when possible
Has tremor improved or resolved?
Yes
No
Enhanced physiologic tremor is the likely diagnosis
This form of tremor is benign and nondisabling
It may recur in states of high adrenergic tone, muscle deconditioning, or with new medication exposure
Are there any neurologic signs other than tremor, such as bradykinesia, altered gait, or ataxia?
Yes
No
Refer to neurology for evaluation; increased likelihood of a combined tremor syndrome
Essential tremor is the likely diagnosis
Neuroimaging is not required in patients with typical clinical features (refer to Table 2)
Refer to UpToDate topic and algorithm on management of essential tremor
Table 1:
associated
Antiarrh
Antidepr
Antiseizu
Beta ago
Glucocor
Lithium
Stimulan
Thyroid
Caffeine
Nicotine
Alcohol
Hyperth
Hypocal
Hyperca
Table 2:
Core crite
Bilateral (but not
Absence
With or lower lin
Secondary
Long du
Positive
Benefici
Exclusion
Isolated
Orthosta
Task- or
Sudden

TSH: thyroid-stimulating hormone; SNRI: serotonin-norepinephrine reuptake inhibitor; SSRI: selective serotonin reuptake inhibitor.

Graphic 140841 Version 1.0

Medications, toxins, and other substances associated with enhanced physiologic tremor

Class	Examples
Antiarrhythmic drugs	Amiodarone
Antidepressant drugs	SNRIs, SSRIs, tricyclics
Antiseizure medications	Bromides, carbamazepine, lacosamide, lamotrigine phenytoin, valproic acid
Beta agonists	Albuterol, terbutaline
Glucocorticoids	Dexamethasone, prednisone
Mood stabilizers	Lithium
Other medications	Cyclosporine, tacrolimus, theophylline
Other substances	Caffeine, cocaine, nicotine
Sympathomimetics	Amphetamine salts, epinephrine, methylphenidate
Thyroid hormone	Levothyroxine
Toxins	Arsenic, cyanide, DDT, lead, lindane, manganese, mercury, naphthalene, toluene

SNRIs: serotonin-norepinephrine reuptake inhibitors; SSRIs: selective serotonin reuptake inhibitors; DDT: dichlorodiphenyltrichloroethane.

Graphic 119320 Version 3.0

Criteria for diagnosis of essential tremor[1,2]

Core criteria	Secondary criteria	Exclusion criteria
▪ Bilateral action tremor of the hands and forearms (but not resting tremor) ▪ Absence of other neurologic signs, with the exception of cogwheel phenomenon ▪ With or without tremor in other locations (head, voice, lower limbs)	▪ Long duration (>3 years) ▪ Positive family history ▪ Beneficial response to alcohol	▪ Isolated focal tremors (voice, head) ▪ Orthostatic tremor with frequency >12 Hz ▪ Task- and position-specific tremors ▪ Sudden onset and stepwise deterioration

References:

1. *Bain P, Brin M, Deuschl G, et al. Criteria for the diagnosis of essential tremor. Neurology 2000; 54(11 Suppl 4):S7.*
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Differentiating Parkinson disease and essential tremor

Clinical features	Parkinson disease tremor	Essential tremor
Age at onset	>50 years	Bimodal 2nd and 6th decade
Sex	Male ≥ Female	Male = Female
Family history	~10 to 15 percent	~50 percent
Asymmetry	+++	+
Frequency	4 to 6 Hz	6 to 12 Hz
Character	At rest	Postural, kinetic
	Supination-pronation	Flexion-extension
Distribution	Hands, legs, chin, tongue	Hands, head, voice
Associated features	Bradykinesia, rigidity, postural instability, micrographia	Mild gait disorder or cerebellar signs in a minority

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Symptoms or signs suggestive of tremor other than essential tremor

Symptom or sign	Likely differential diagnosis
Unilateral tremor, leg tremor, rigidity, bradykinesia, rest tremor	Parkinson disease
Gait disturbance	Parkinson disease; cerebellar tremor
Irregular, jerky tremor	Dystonic tremor
Isolated head tremor with abnormal posture (head tilt or turning)	Dystonic tremor
Sudden or rapid onset	Functional (psychogenic) tremor; toxic tremor
Current drug treatment that may cause or exacerbate tremor	Drug-induced or toxic tremor

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Differential diagnosis of tremor

Tremor	Description
Essential tremor	Bilateral postural or kinetic tremor of the hands and forearms (≥4 Hz; usually 6 to 12 Hz) or isolated head tremor without evidence of dystonia. Absence of other neurologic signs or recent trauma preceding the onset of tremor. Can be asymmetric.
Physiologic tremor	Enhanced physiologic tremor. High frequency (10 to 12 Hz), presence of known cause (eg, medications, hyperthyroidism, hypoglycemia).
Parkinson disease	Rest tremor, unilateral at onset. Can be mixture of rest and action tremors; occasionally action tremor alone. Asymmetry sustained when bilateral. Leg or foot tremor more common than with essential tremor, usually does not produce head tremor. Frequency 4 to 6 Hz.
Orthostatic tremor	Postural tremor in the torso and lower limbs while standing; may also occur in the upper limbs. Suppressed by walking. Tremor is high frequency (14 to 20 Hz) and synchronous among ipsilateral and contralateral muscles.
Cerebellar tremor	Postural, intention, or action tremor. Relatively low frequency (3 to 4 Hz). Associated with ataxia and dysmetria.
Neuropathic tremor	Variable tremor type and frequency, usually postural and kinetic tremor in the involved extremities. Other signs of peripheral neuropathy present.
Rubral or midbrain tremor	Mixture of rest, postural, and intention tremor with frequency of 2 to 5 Hz. Always associated with signs of brainstem or cerebellar damage.
Dystonic tremor	Action tremor, usually postural or task-specific, in a body part that is affected by dystonia. Usually slow, irregular, and jerky. Common examples are head and/or arm tremor due to cervical dystonia. May be responsive to alleviating maneuvers (sensory trick).
Functional tremor	Can affect any body part with rest, action, or both. Variable in direction, amplitude, and frequency. Distractible and entrainable. When physically suppressed by examiner, tremor may appear in another limb.

Data from:

1. *Elble RJ. Diagnostic criteria for essential tremor and differential diagnosis. Neurology 2000; 54(11 Suppl 4):S2.*
2. *Louis ED. Essential tremors: a family of neurodegenerative disorders? Arch Neurol 2009; 66:1202.*
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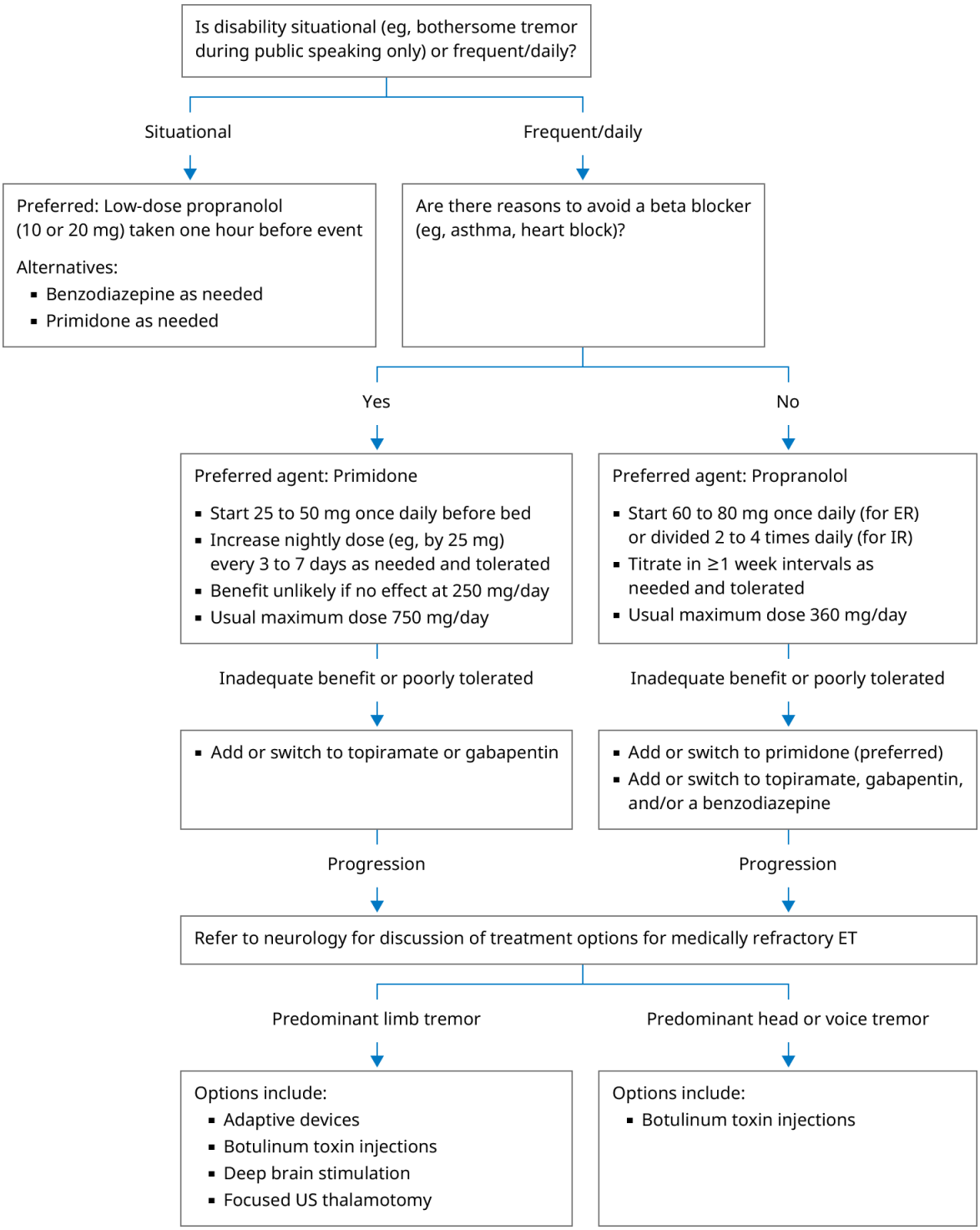
Graphic 77497 Version 6.0

Symptomatic management of essential tremor (ET) in adults


Is disability situational (eg, bothersome tremor during public speaking only) or frequent/daily?
Situational
Frequent/daily
Preferred: Low-dose propranolol (10 or 20 mg) taken one hour before event
Alternatives:
Benzodiazepine as needed
Primidone as needed
Are there reasons to avoid a beta blocker (eg, asthma, heart block)?
Yes
No
Preferred agent: Primidone
Start 25 to 50 mg once daily before bed
Increase nightly dose (eg, by 25 mg) every 3 to 7 days as needed and tolerated
Benefit unlikely if no effect at 250 mg/day
Usual maximum dose 750 mg/day
Preferred agent: Propranolol
Start 60 to 80 mg once daily (for ER) or divided 2 to 4 times daily (for IR)
Titrate in ≥1 week intervals as needed and tolerated
Usual maximum dose 360 mg/day
Inadequate benefit or poorly tolerated
Inadequate benefit or poorly tolerated
Add or switch to topiramate or gabapentin
Add or switch to primidone (preferred)
Add or switch to topiramate, gabapentin, and/or a benzodiazepine
Progression
Progression
Refer to neurology for discussion of treatment options for medically refractory ET
Predominant limb tremor
Predominant head or voice tremor
Options include:
Adaptive devices
Botulinum toxin injections
Deep brain stimulation
Focused US thalamotomy
Options include:
Botulinum toxin injections

Management of essential tremor (ET) is symptomatic. The goal of pharmacotherapy is to reduce tremor severity enough to mitigate disability while minimizing side effects. Before starting medication, assess tremor severity, distribution (limb, head, and/or voice), impact on activities of daily living, and degree of functional and social disability. Inquire about common fears and concerns (eg, Parkinson disease). Review and eliminate exacerbating factors, when possible. Dosing in this algorithm is for oral administration in adults with normal kidney function.

ER: extended-release; IR: immediate-release; US: ultrasound.

Graphic 104216 Version 7.0

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